factors, possibly immunological, must be involved in determining the clinical response to infection.

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REFERENCES

Maternal histidinaemia

Sir,

Recently two questions have been raised concerning the inherited biochemical trait histidinaemia. (1) Is the trait a benign metabolic disorder (Popkin et al., 1974; Levy, Shih, and Madigan, 1974)? (2) Are children born of histidinaemic mothers at risk of harm during gestation (Neville et al., 1971; Lyon, Gardner, and Veale, 1974)? The answers to these questions are important, since the unnecessary administration of a histidine-deficient diet to pregnant women or to young infants with biochemical histidinaemia might result in a harmful nutritional imbalance. We report a normal histidinaemic mother with 4 normal children.

Plasma and urine amino acids were measured with an amino acid analyser. Imidazolepyruvic acid was determined spectrophotometrically by the borate-arsonate method (Lin et al., 1958). Imidazololactic acid, imidazoleacetic acid, and N-acetylhistidine were estimated by visual comparison of colour produced by diazotized sulphanilic acid with materials in aliquots of urine and with known amounts of authentic compounds after two-dimensional paper chromatography on Whatman No. 3MM filter paper in pyridine-acetone-3 N ammonium hydroxide (50:30:25), followed by isopropyl alcohol-formic acid (88%-water (8:1:1).

The mother was on our staff; her plasma amino acids were measured during collection of data on plasma amino acid levels of normal children and adults. Biochemically she had typical histidinaemia. Her fasting plasma histidine levels were 0.73, 0.52, and 0.79 μmol/ml in 3 samples collected over a 6-month period.

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REFERENCES

Dr. Wise and colleagues comment as follows:
We agree entirely with the comments of Dr. Buckler. Since submission of our original paper, and in an attempt to provide a more consistently effective stimulus, we have modified our exercise test to a 10- to 20-minute period (depending on endurance) workload of 450 kilopond [(4410 N) m/min using a bicycle ergometer (Monark). Samples for growth hormone assay were taken at 1 and 15 minutes after completion of exercise, so that a minimum interval of 25 minutes from the start of exercise to final sampling applied to all cases. Over 90% of 24 subjects with short stature, and who were not growth-hormone deficient exceeded 14 μU/ml in one or both of these samples.

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Exercise as a physiological stimulus to growth hormone release

Sir,

In comparing methods of stimulating growth hormone release, Wise et al. (1975) included exercise, but reported that levels exceeding 14 μU/ml were only obtained in 50% of cases who were not growth-hormone deficient. The technique they described involved blood sampling 10 minutes after completion of exercise to mild exhaustion by stair climbing. Extensive studies on several subjects (Buckler, 1972) have shown that plasma growth hormone levels associated with exercise do not begin to rise for 10–15 minutes and do not reach peak values until 25–30 minutes after the start of exercise. Unless the children in the report by Wise et al. climbed stairs for at least 15 minutes, it is unlikely that the greatest response in plasma growth hormone levels would be detected by their method. It is possible that others who report poor responses with this method of exercise testing may have failed to take the blood sample at the correct time, namely 25–30 minutes after the start of a short (5–10 minute) period of intensive exercise.

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